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SUMMARY OF CRITICISMS ON EPA RISK ASSESSMENT

The EPA Risk Assessment includes a selective review of the literature on mainstream smoke, coupled with a series of unfounded and demonstrably false assumptions.

- The Risk Assessment's "hazard identification" does not review published data on the physical and chemical characteristics of ETS and relies upon the assumption that mainstream smoke and ETS are similar substances.

- The EPA document fails to recognize that the tobacco smoke to which the nonsmoker is exposed differs both physically and quantitatively from mainstream smoke.[1-8]

  The Risk Assessment does not address these differences and does not attempt any chemical or quantitative comparison between ETS and mainstream smoke.

  ETS is a highly diluted, aged and chemically altered mixture of sidestream smoke (the smoke emitted from the burning end of the cigarette) and exhaled mainstream smoke. The chemical composition of this mixture changes as it ages and interacts with other materials present in the room air.

- The active smoking data presented by the EPA are not relevant to the suspected route of exposure for ETS or, given the

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physical and quantitative differences between mainstream smoke and ETS, to a proper characterization of ETS itself.

Indeed, previous scientific reviews of ETS, including reviews by the U.S. Surgeon General[9] and the National Academy of Sciences,[10] have cautioned against such comparisons, stressing that there are significant differences between ETS and mainstream smoke that make assumptions of equivalence scientifically unjustified.

EPA does not adequately discuss the available data from animal studies of ETS exposure.

- The only animal data referred to in the Risk Assessment are from a single inhalation study on hamsters in which it was reported that animals exposed to mainstream smoke exhibited an increase in laryngeal tumors; several studies on lung implantation of tobacco smoke condensates; and several results from studies of mouse skin-painting of cigarette smoke condensates.

- Studies relating to laryngeal cancer and to the reported results from implantation or skin-painting studies have little relevance to either the expected route of exposure for ETS,

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i.e., inhalation to the lung, or to the disease endpoint under discussion (lung cancer).

- Animal inhalation studies employing fresh sidestream smoke and ETS were similarly not discussed in the Risk Assessment.

  All three publicly available studies to date have reported no statistically significant increased incidence of tumors in animals exposed to ETS compared with controls.[11-14]

  Moreover, the Risk Assessment fails to recognize that none of the lifetime, "whole smoke" exposure studies on animals, originally designed to assess possible biological effects from mainstream smoke exposures, have reported the induction of lung carcinomas via inhalation.

The Risk Assessment fails to reference a number of actual studies comparing levels of mutagens and other genotoxic markers in the body fluids of exposed and non-exposed nonsmokers.[15-24]

- The results of those studies suggest no statistically significant increases in mutagenic activity in the body fluids of nonsmokers exposed to realistic levels of ETS compared with nonsmokers who are not exposed.

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- Moreover, the "carcinogenic agents" supposedly identified in tobacco smoke (e.g., the "list" of suspected carcinogens referred to in the Risk Assessment) either are not suspected pulmonary carcinogens or have not been unequivocally demonstrated as tumorigenic to human tissue or to the lung tissue of experimental animals.[25-27]

In summary, the Risk Assessment's failure to include and consider relevant animal and short-term test data on ETS renders its "hazard identification" section incomplete. The active smoking data presented in the chapter are not relevant to the suspected route of exposure for ETS or, given the physical and chemical differences between MS and ETS, to a proper characterization of ETS itself. In addition, experimental inhalation data on MS, SS and ETS in animal models, not addressed in the chapter, provide no support for the claim that ETS is causally related to an increased risk of lung cancer.

In order to obtain the conclusion of the Risk Assessment, namely a statistically significant estimate of risk, the EPA combined data from epidemiologic studies using a statistical technique known as meta-analysis. The EPA's meta-analysis is scientifically unsound for a number of reasons.

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- The EPA's meta-analysis violated a fundamental principle of meta-analysis: it did not aggregate similar data. For example, all of the studies employed different questionnaires and different assessments of past exposure; five of the studies failed to match cases with controls; and the criteria for control selection were different in each study.[28]

- If the EPA had sought to address the lack of statistical power in some of the studies, it could have placed less emphasis on those studies and more upon the individual studies with sufficient statistical power. The individual studies with sufficient power do not report overall point estimates that achieve statistical significance.

- The statistical power question notwithstanding, the EPA has not addressed the issue of the aggregation of dissimilar data in its meta-analysis. Moreover, even the "common thread" identified by the EPA as justification for meta-analysis, spousal smoking, is defined differently across studies.

The EPA relied on a cumulative risk estimate derived from the meta-analysis of 11 U.S. epidemiologic studies of spousal smoking for its classification of ETS as a known human carcinogen.[29-39]

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- Not one of those 11 studies originally reported an overall risk estimate for lung cancer that was statistically significant, i.e., chance was not effectively ruled out as an explanation for the reported association between spousal smoking and lung cancer in nonsmokers.

- Indeed, even when the EPA lowered the statistical confidence limits reported for the U.S. studies from the standard 95% to 90%, all but one of the 11 studies individually failed to achieve statistical significance.[33]

- The EPA also reviewed 19 other epidemiologic studies from seven countries other than the United States.[40-58] Of these 19 studies, 13 reported no statistically significant overall point estimate for spousal smoking and lung cancer.

- Put another way, of the 30 studies reviewed by the EPA in all, 24 (a full 80%) did not support the Agency's conclusion. How then, can the EPA justify its conclusion that the epidemiology supports the classification of ETS as a Group A carcinogen?

- Even positive point estimates (greater than 1.00) that do not achieve statistical significance are compatible with the null hypothesis of "no association" between spousal smoking and

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lung cancer. Reported overall point estimates in six non-U.S. studies are statistically significant, but the EPA did not ascertain whether or not these purported associations are due to some feature that is coincidental to "marriage to a smoker" (e.g., common diet, occupational exposures, socio-economic status and other lifestyle characteristics).

The EPA intentionally lowered the confidence interval reported in the individual epidemiologic studies from 95% to 90% in an apparent attempt to rule out chance, and thus obtain a statistically significant cumulative risk estimate in its meta-analysis of U.S. studies.

- A confidence interval measures the probability that a statistical association was obtained by chance. The net effect of lowering the confidence interval from 95% to 90% is to artificially double the likelihood of ruling out chance as a possible explanation.

- The EPA's use of a lower confidence interval cannot be justified scientifically in light of the following obervations:

  The EPA and other federal agencies have generally used 95% confidence intervals in other risk assessments that have relied upon epidemiologic studies.

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Virtually all of the ETS epidemiologic studies upon which the EPA relied originally reported 95% confidence intervals.

The EPA's 1990 draft risk assessment on ETS used 95% confidence intervals.

Lowering the confidence interval was apparently the only way the EPA could reach its conclusion of a statistically significant increased risk for the combined U.S. studies.

The ETS Risk Assessment does not discuss the effect that lowering the confidence interval had on the data.

- The magnitude of the effect of the EPA's manipulation of the data is revealed when the standard 95% confidence interval is used. A meta-analysis of the U.S. studies employing the EPA's assumptions and using a 95% confidence interval reports no statistically significant summary risk estimate.[59]

The EPA failed to include in its meta-analysis relevant data from two major studies on ETS.[60,61] Months prior to the release of the Risk Assessment, these two studies appeared in the scientific literature, examining the purported association between spousal smoking status and lung cancer.

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- Funded in part by the National Cancer Institute, the 1992 Brownson, et al., study is one of the largest studies ever conducted on ETS exposure and lung cancer incidence.[60] It was not included in the Risk Assessment.

- The EPA had access to the two studies well in advance of the release of the final Risk Assessment.

- If the two studies were included in the EPA's meta-analysis of U.S. studies, a statistically nonsignificant cumulative risk estimate for spousal smoking would be calculated, even at the 90% confidence interval used by the EPA.[59]

- The inclusion of both the studies in the meta-analysis should negate the classification of ETS as a Group A carcinogen, in that a cumulative risk estimate for the U.S. studies would not be statistically significant, even at the 90% confidence interval.[59] The Agency should have estimated no statistically significant increased risk for lung cancer, at either a 95% or a 90% confidence interval.

- A meta-analysis including the two recent studies was forwarded to the EPA more than one month prior to the release of the final Risk Assessment.[59] The EPA chose not to include those

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data in its statistical calculations in the final Risk Assessment.[62]

The EPA reported a cumulative point estimate via meta-analysis for ETS of 1.19, when a risk estimate of 1.0 indicates no increase or decrease in reported risk. Epidemiologists generally agree that any estimated risk of less than 2.0 should be deemed "weak."[63]

- A point estimate below 2.0, given the very nature of epidemiology, may be due to some factor or factors associated with marriage to a smoker (e.g., diet, lifestyle, occupation, socioeconomic status, etc.).[64-74]

- Epidemiologic studies have reported risk estimates for lung cancer in nonsmokers in excess of 2.0 for these factors. The scientific literature suggests that such estimates may be large enough to account completely for the reported association between spousal smoking status and lung cancer.

- The EPA did not address fundamental questions regarding the detection limits of an epidemiologic study, or whether confounding factors have been effectively ruled out.

Only six of the 30 epidemiologic studies considered by the EPA reported overall statistically significant associations between

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spousal smoking and lung cancer; moreover, all six were conducted outside the United States.[43,45,47,49,50,57]

- These studies, as noted above for the U.S. studies, employed inconsistent methods of design and analysis.

- More importantly, the EPA failed to investigate whether the statistically significant associations reported in those studies could be the result of confounding by potential risk factors. The EPA failed to consider the large body of literature independent of the spousal smoking studies that addresses such factors.[64-87]

- EPA took the position that it was necessary to identify a single confounder, applicable across all the studies. Given the disparate study populations, methods, and analysis employed in the 30 studies, the identification of a single confounder to explain the reported association between spousal smoking and lung cancer is an unwarranted and, indeed, an unscientific expectation.[74]

Moreover, the scientific literature identifies several sources of possible bias in the spousal smoking studies.[88-98] These include publication bias, recall (exposure) bias, disease misclassification and smoking status misclassification.

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The ETS Risk Assessment itself states (3-53) that "misclassification errors must be addressed when using questionnaires to assess ETS exposure." The statement refers to exposure misclassification bias, which, along with other potential sources of bias, was not adequately addressed in the Risk Assessment.

- The Risk Assessment acknowledges only <u>one</u> possible bias, smoking status misclassification, and adjusts for it by employing an unpublished scientific model that had not been subjected to external peer review.

  The model contains numerous mathematical and conceptual errors, including a misclassification rate that is not representative of the U.S. population.[99-100]

  If a realistic misclassification rate had been used, EPA's own meta-analysis would have reported no statistically significant association between spousal smoking status and lung cancer.

- Adjustments for misclassification and "background exposure" are not meaningful, insofar as these adjustments were not performed by the original authors on data from their respective studies.

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The "background adjustment" is questionable since it relies on selected exposure data.

Moreover, the background adjustment presumes a causal relation for spousal smoking and lung cancer.

In relying on the 30 spousal smoking studies, the EPA selectively chose which data to use. Specifically, the EPA limited examination to the reported association between spousal smoking status in females and lung cancer.

- Although 11 of the 30 epidemiologic studies considered by the EPA also assessed workplace exposures to ETS, the Risk Assessment did not address those data.[31,33,35,37-39,47,48,51,54,58]

  Nine of the 11 spousal smoking studies that examined workplace exposures reported no statistically significant association with lung cancer.[31,35,37,39,47,48,51,54,58]

  If the data on workplace exposure reported in the 11 studies are pooled in a meta-analysis such as the one conducted in the Risk Assessment, the summary risk estimate is not statistically significant.[101]

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- Similarly, ten of the 30 epidemiologic studies contained data regarding reported exposures to ETS during childhood. The Risk Assessment also failed to consider those data.[32-34,37,39,40,42,48,55,56]

  Nine of the ten studies examining reported exposures to ETS during childhood reported no statistically significant association for lung cancer in adult nonsmokers.[32-34,39,40,42,48,55,56]

- The failure of the EPA to consider those data was scientifically unjustified. If the EPA had reviewed the data on workplace exposure and childhood residential exposure, the Agency should have found that the data contradicted its own conclusions.

Reports on dioxin, diesel emissions and EMF, prepared by working groups of EPA's Science Advisory Board, rejected the use of epidemiologic data like those in the ETS risk assessment precisely because the reported risks were low, and because the epidemiologic studies were not based on actual exposure data.

- The SAB's Ad Hoc Panel on Dioxin (November 1989) criticized a Review Draft on dioxin for just those oversights.[102]

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The SAB Report noted that most of the epidemiologic studies evaluated for dioxin "do not provide definitive data" and should be classified as "inconclusive, due in most cases to design limitations such as inadequate power and inadequate exposure assessment."

The Report concluded that "without good exposure data, the epidemiologic studies are meaningless."

- The SAB Workshop Review Draft on diesel emissions stated:[103]

> An excess risk of lung cancer was observed in three out of seven cohort studies and six out of seven case-control studies. Of these studies, two cohort and two case-control studies observed a dose-response relationship using duration of employment as a surrogate for dose. However, due to the lack of actual data on exposure to diesel exhaust in these studies and other methodologic limitations such as lack of latency analysis etc., the evidence of carcinogenicity in humans is considered to be limited for diesel exhaust exposure. (emphasis added)

The SAB Working Group report on EMF observed:[104]

> The association between cancer occurrence and exposure to either ELF or RF fields is not strong enough to constitute a proven causal relationship, largely because the relative risks in the published reports have seldom exceeded 3.0 in both childhood residential exposures and in occupational situations. (emphasis added)

And:

> The consistently repeated pattern of lymphoma, leukemia, nervous system cancer and lymphoma in childhood studies and the ruling out of several confounding exposure factors in the Savitz, et al. (1988) study argue in favor of a causal link between these tumor types in children and exposure to ELF magnetic or electric fields. However,

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the fact that the odds ratios are small and in many cases not statistically significant indicates that the association may not be strong and therefore argues against a causal relationship. (emphasis added)

A central contention of the Risk Assessment is that purported similarities between active smoking (mainstream smoke) and nonsmoker exposure to ETS imply "biologic plausibility" of the claim that ETS is a Group A carcinogen.

- Of course, the argument by analogy from active smoking to the "biologic plausibility" of the alleged role of ETS in disease causation does not provide a scientific evaluation of the hypothesis on ETS. Instead, it simply suggests the compatibility of two hypotheses, one for active smoking and one for ETS, in relation to human disease.

- The "biologic plausibility" question should be addressed by reference to animal inhalation studies on ETS and short-term tests on humans for ETS.

  The published results from these studies are negative for ETS.

The EPA's "dose-response analysis" is limited to performing a "re-analysis" of exposure-response trends reported in the individual spousal smoking studies.

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- Data are presented in some of the spousal studies in such a way that permits comparison of exposure data (i.e., the number of cigarettes or duration of recalled exposure) with estimates of risk. Such data are amenable to what statisticians refer to as a "trend test."

- The EPA's analysis did not rely on the study data as reported. Instead, the authors of the Risk Assessment recalculated trends for dose-response by adding what statisticians call a "pseudo-datum" representing zero exposure.[105]

- The addition of the "pseudo-datum" converts the statistically non-significant dose-response trends originally reported in the individual studies into statistically significant trends.

- The use of this inappropriate procedure essentially permits the establishment of a dose-response trend based upon any single category of exposure greater than zero.[105]

- Such trends do not support the existence of a dose-response among exposed individuals, nor do they rule out the possibility that the claimed associations relied upon are attributable to confounding or other factors that are correlated with spousal smoking.

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- The EPA suggests that its trend tests using unexposed subjects provide "evidential support of a causal association." However, if the dose-response analysis is performed correctly (excluding unexposed subjects), no spousal smoking study reports a significant dose-response trend.[105,106]

The ETS Risk Assessment is not based on accurate or verifiable information concerning exposure to ETS.

- The EPA relied upon questionnaire data on exposure recall contained in the spousal smoking studies.

- The accuracy of that data depends on an individual's ability to recall past events, such as how much a husband smoked in the past 20 to 30 years.[88,89,92]

The Risk Assessment fails to discuss or reference much of the relevant literature on the physical and chemical properties of ETS (as a mixture distinct from mainstream or sidestream smoke).

- In addition, only a small number of actual ETS exposure studies available in the published literature are discussed, and none of these are integrated into the body of the Risk Assessment.

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- Many of the studies of exposure to ETS constituents under realistic conditions in public places, workplaces and homes omitted from the Risk Assessment report minimal exposures to ETS: these reported exposures do not support the conclusions of the Risk Assessment.[1,4-8,107-138]

- The initial public review draft for the ETS Risk Assessment (1990) did not even contain an exposure assessment.

  Comments submitted to the public docket for the 1990 draft risk assessment observed that the EPA had failed to provide an exposure assessment which considered data from the numerous published studies on actual levels of ETS constituents in the air of public places and workplaces.

- In apparent response to that criticism, the revised 1992 draft of the ETS Risk Assessment contained a chapter entitled "Estimation of Environmental Tobacco Smoke Exposure," but the chapter's authors failed to consider at least 35 pertinent exposure studies on ETS constituent levels in public places.

  Furthermore, during its review of the chapter in July of 1992, the EPA's Science Advisory Board rejected the chapter and returned it to the author for rewriting.

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Nevertheless, without either an exposure assessment or recourse to any of the data pertaining to it, the Science Advisory Board endorsed the EPA's estimate of exposure and risk for the entire U.S. population.

A revised chapter on ETS exposure occurs in the final ETS Risk Assessment; however, the studies and data therein are not integrated into the Risk Assessment.

While some reports suggest that cotinine is a reliable marker for exposure to tobacco smoke,[139-140] critical assessments demonstrate that cotinine cannot serve as an accurate measure of ETS exposure.[141-150]

- It has been reported that individuals metabolize nicotine in different ways at different times and that elimination rates for cotinine vary among individuals.

- In addition, recent research suggests that diet may contribute to levels of nicotine and cotinine found in the body, thereby interfering with reported exposure levels from nicotine in the ambient air.[151]

- Scientists have also noted that different methods of analysis may influence final recorded levels of cotinine.[152]

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- Finally, it has been reported that because nicotine is largely present in the gas phase of ETS, measurement levels of its metabolite, cotinine, do not reflect exposures to other constituents that may be present, for example, in the particulate phase of ETS.

- For these reasons, cotinine should not be regarded as a reliable quantitative measure of ETS exposure.

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